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DATE: Thursday, August 11, 2005

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		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=OR	
☐	L1	(Interferon adj alpha) with (HBsAg or HBeAg)	10

END OF SEARCH HISTORY

1: Gut. 1993;34(2 Suppl):S87-90. Related Articles, Links

Interferon alfa-2b therapy in children with chronic hepatitis B.

Sokal EM, Wirth S, Goyens P, Depreterre A, Cornu C.

Department of Paediatrics, Universite Catholique de Louvain, Hopital Universitaire des Enfants (ULB), Brussels.

Twenty nine children (mean age 8.3 years, 18 boys, 11 girls) who had biopsy proved chronic hepatitis B virus infection (HBV) with active viral replication were given a 16 week course of interferon alfa-2b treatment (9 million units (MU)/m2, thrice weekly). Fourteen children (48%) showed persistent loss of HBV-DNA 8 months after the end of treatment; 11 (38%) lost hepatitis B e antigen (HBeAg), and two (7%) hepatitis B surface antigen (HBsAg). Alanine aminotransferase activities returned to normal in 12 children. Those who responded had significantly higher initial transaminase activities than those who did not ($p < 0.01$) but similar serum HBV-DNA. Results were compared with the natural evolution of the disease in a group of 25 children (mean age 8.3 years) with identical initial mean serum HBV-DNA values, followed up during the same period. Two of these (8%) lost HBeAg and one (4%) HBsAg. The 23 remaining control subjects had no decrease in serum HBV-DNA or in transaminase activities compared with values 1 year earlier. It is concluded that treatment with interferon alfa-2b in children may lead to inhibition of HBV replication similar to that described in adults, and may thus shorten disease evolution. Further studies are necessary to establish the best protocols and to identify those patients who are the most likely to respond to treatment.

Publication Types:
- Clinical Trial
- Multicenter Study

PMID: 8314496 [PubMed - indexed for MEDLINE]

1: J Interferon Res. 1992 Apr;12(2):139-43.

Engineered disulfide bond greatly increases specific activity of recombinant murine interferon-beta.

Day C, Schwartz B, Li BL, Pestka S.

National Institutes of Health, Institute of Allergies & Infectious Diseases, Rockville, MD 20852.

Unlike other species of interferon-beta (IFN-beta) mouse (Mu) IFN-beta has no naturally occurring intramolecular disulfide bond. When expressed in Escherichia coli, MuIFN-beta appears to exhibit instability and low activity. To increase its activity, we engineered a pair of cysteines into recombinant MuIFN-beta to test whether this change would improve its antiviral activity. In the absence of detailed structural data, the optimal placement of cysteines was determined by sequence comparison with other species of IFN-beta. While MuIFN-beta has only a single cysteine (at position 17), other species of IFN-beta have three cysteines, at positions 17, 31, and 141 (numbering based on consensus sequence), a disulfide bond being formed between the latter two residues. Thus, we introduced cysteines into MuIFN-beta at positions 29 and 136, which correspond to positions 31 and 141 of the consensus sequence. When the variant form of MuIFN-beta was expressed in bacteria and purified, we found that the additional cysteines greatly increased the antiviral activity. Further improvement was obtained by replacement of the Cys-17 with a serine. In this manner a specific activity approximately 15-fold that of the wild-type recombinant molecule was achieved.

PMID: 1578187 [PubMed - indexed for MEDLINE]

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=> s l1 (w) inhibi?
L2 356 L1 (W) INHIBI?

=> s l2 (s) (HBsAg or HBeAg)
L3 4 L2 (S) (HBSAG OR HBEAG)

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AB Efficacy and safety of therapy with lymphoblastoid interferon-alpha alone or combined with deflazacort has been investigated in 38 HBsAg-HBeAg+ patients with biopsy-proven chronic hepatitis. Group I received 5 MU/m2 interferon thrice a week for 26 weeks; group II took interferon for 26 weeks simultaneously with a 6-week course of deflazacort. Follow-up was 18-72 months (median 42). After 12 months, responses were achieved in 3 (18%) out of 17 patients on interferon alone vs 5 (26%, p > 0.05) out of 19 on combined therapy. Blind histological assessment revealed no improvement in either group or in patients who responded to therapy within the first year of follow-up ("early responders"). "Delayed" responses were observed in 4 (29%) patients who took interferon alone vs 5 (36%, p > 0.05) who took the combined therapy. Serum HBV DNA levels decreased significantly during treatment and remained low up to 24 and 36 months of follow-up in both groups. One early responder developed hepatocellular carcinoma, another had exacerbation of liver disease in long-term follow-up. No non-responders developed liver failure or hepatocellular carcinoma. These results indicate that lymphoblastoid **interferon -alpha inhibits** HBV replication and corticosteroids have no synergistic effect in treatment of **HBsAg-HBeAg** + chronic hepatitis.

ACCESSION NUMBER: 97046926 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8891847
TITLE: Treatment of chronic hepatitis B (HBeAg-HBV DNA-positive) with lymphoblastoid alpha interferon with or without corticosteroids: short- and long-term follow-up.
AUTHOR: Zavaglia C; Bottelli R; Bellati G; Asti L; Mondazzi L; Iamoni G; Zanetti A; Tanzi E; Fesce E; Gelosa F; Maggi G; Ideo G
CORPORATE SOURCE: Divisione di Medicina Generale e Servizio di Epatologia, Ospedale Niguarda, Milano, Italy.
SOURCE: Italian journal of gastroenterology, (1996 Jul-Aug) 28 (6) 324-31.
Journal code: 8000544. ISSN: 0392-0623.
PUB. COUNTRY: Italy
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19970219
Entered Medline: 19970131

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